Psychiatria Danubina, 2024; Vol. 36, No. 2, pp 225-231 https://doi.org/10.24869/psyd.2024.217 *Original research*

INVESTIGATION OF THE EFFECT OF THE ONSET OF BRUXISM AS A RESULT OF EARLY ANTIDEPRESSANT USE

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received: 31. 1. 2023 ; revised: 12. 11. 2023; accepted: 15. 11. 2023

Summary

Background: *Among antidepressants, selective serotonin and noradrenaline reuptake inhibitors (SSRIs and SNRIs) have been widely used in the treatment of major depression and may induce sleep disorders and bruxism. In the present study, the effects of SSRIs and SNRIs on awake and sleep bruxism have been evaluated.*

Subjects and methods: *A total of 125 patients who had been prescribed SSRIs or SNRIs for the treatment of major depression have been evaluated for bruxism. For the purpose of the study, data from the first week (T1) and the fourth week (T2) of antidepressant treatment have been considered.*

Results: *In conclusion, in the early period, the presence of bruxism has not been observed to be significantly influenced by the use of antidepressants. It has been determined that sleep bruxism increased in the fourth week only in males who were using antidepressants ($p = 0.015$; $p < 0.05$). An increase in the presence of sleep bruxism due to specific SSRIs and SNRIs has been determined in the fourth week of drug use. Paroxetine in the SSRI group and duloxetine in the SNRI group have been found to cause an increase in sleep bruxism ($p = 0.013$; $p < 0.05$). Other active substances have not been found to affect sleep or awake bruxism significantly.*

Conclusion: *The present study has shown that although some antidepressants increase bruxism in the early period of drug use, the effects of similar drugs on sleep or awake bruxism need to be evaluated in detail in long-term studies.*

Keywords: *bruxism, depression, antidepressant, SSRI, SNRI*

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INTRODUCTION

Bruxism was first reported in the early 1900s. The word comes from the Greek word "brgymos", which means clenching teeth (Castrillon and Exposto 2018). Since then, there have been various definitions over the years. More recently, an international consensus has accepted a simple explanation of bruxism, which is a repetitive masticatory muscle activity characterized by clenching or grinding of the teeth and support or pushing of the mandible. According to this consensus, bruxism can be classified as sleep bruxism or awake bruxism, depending on the circadian phenotype of the affected individual (Lobbezoo et al. 2018). The etiology of bruxism includes psychological factors, trauma, genetic predisposition, tobacco intake, caffeine consumption, some narcotic drugs, and drugs such as selective serotonin and noradrenaline reuptake inhibitors (SSRIs and SNRIs) that can lead to sleep disorders (Yagci et al. 2019). Bruxism is known to cause dental erosion, temporomandibular joint disorders (TMDs) and pain in the muscles used for mastication.

While the severity of bruxism varies between individuals, the symptoms are generally similar. These include pain in the masticatory system, mandibular fatigue, and decreased mouth opening. There is no widely accepted specific form for diagnosing bruxism. The Axis-I section of the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) form is generally used in the diagnosis of TMDs, and the "Oral Habits Checklist", which is part of the Axis-II section, is used in the diagnosis of parafunctional habits associated with TMDs (Ohrbach and Dworkin 2016). Since the DC/TMD form is a tool for diagnosing all TMDs, it is not practical to use it only to diagnose bruxism. Therefore, the diagnosis of bruxism is often based on clinical symptoms, self-awareness, and feedback from individuals themselves or their sleep partners, rather than using tools such as polysomnography (PSG) or electromyography (EMG) (Miettinen et al. 2020). Diagnostic forms and questionnaires can diagnose bruxism with questions about clinical symptoms (Winocur et al. 2011).

Serotonin and noradrenaline reuptake inhibitors (SSRIs and SNRIs) might be used for several psychological conditions such as major depression, obsessive-compulsive, anxiety, post-traumatic, and bipolar disorders. They are taken orally and are easily tolerated; hence they are widely prescribed for adolescents and adults (DeLucia et al. 2016). SSRIs and SNRIs block serotonin and noradrenalin

release, causing these substances to accumulate in the synaptic space. Therefore, these substances remain longer in blood circulation (Xue et al. 2016). Patients may suffer from some adverse effects, including weight changes, loss of libido, sleep problems, gastrointestinal dysfunction, and headache (DeLucia et al. 2016). Symptoms may begin within 2 to 4 weeks after drug initiation (Rajan and Sun 2017; Sabuncuoglu et al. 2009; Garrett and Hawley 2018). Several antidepressants might worsen or trigger primary sleep disorders such as restless legs syndrome, sleep bruxism, REM sleep behaviour disorder, nightmares, and sleep apnea. It has been reported that SSRIs and SNRIs cause sleep bruxism (Albayrak and Ekinci 2011; Garrett and Hawley 2018; Uca et al. 2015). Some studies have classified bruxism as "primary" if the underlying condition cannot be identified conclusively and ''secondary'' if it occurs following drug use (Melo et al. 2018). The study's authors used "secondary" identification to mention drug (SSRIs and SNRIs))-induced bruxism.

The focus on the etiology of bruxism has generally shifted from environmental factors affecting the peripheral nervous system (PNS) to pathophysiological factors affecting the central nervous system (CNS) (Klasser et al. 2015). It has been reported that sleep bruxism can be triggered by neurotransmitters such as dopamine that regulate the CNS (Lobbezoo et al. 1997; Lavigne et al. 2001). The first evidence of bruxism associated with dopamine release is a case report of the treatment of a patient with Parkinson's disease using 1-3,4-dihydroxyphenylalanine (L-dopa) (Winocur et al. 2011). In several controlled studies on young and healthy patients with symptoms of sleep bruxism, it was reported that the use of L-dopa caused a significant reduction in bruxism activity and severity of bruxism during RMMA (Rhythmic Mimicatory Muscle Activity) compared to placebo (Lavigne et al. 2001). The basal ganglia are parallel designed functional components consisting of the thalamus and cortex, and the information flow within them controls muscle movements by organizing the preparatory process of motor functions (Alexander and Crutcher 1990). Based on the CNS, it is thought that disorders in the basal ganglia (caudate nucleus, putamen, globus pallidus), such as sleep dysfunctions, may cause bruxism. The overall effect of dopamine on the striatum may occur by potentiating an activation induced by the cortical pathway or by directly affecting the circuit that has an excitatory effect on the thalamus, thereby accelerating transmission (Behr et al. 2012). Serotonin (5-HT2) blockade theoretically reduces dopaminergic neuron inhibition and causes an indirect dopaminergic effect. It is thought that SSRI or SNRI use may lead to an increase in mesocortical dopamine release and bruxism intensity (Romanelli et al. 1996).

In order to contribute to and shed light on the studies in the literature, this study aimed to examine the relationship between awake and sleep bruxism observed in patients using SSRI and SNRI group antidepressants.

SUBJECTS AND METHODS

This prospective cross-sectional pilot study design and reporting are based on the STROBE guidelines (Strengthening the Reporting of Observational Studies in Epidemiology) Statement: Guidelines for Reporting Observational Studies, 2014). On the other hand, the study has been planned to be conducted with a total of 175 patients who were prescribed antidepressants following the diagnosis of *major depressive disorder (MDD*) at Kocaeli University, School of Medicine, department of Psychiatry, between November 2019 and March 2020. Since the main purpose of the study was to be conducted at Istanbul Medipol University, the necessary correspondence between the institutions to conduct the study with the data of the patients who applied to Kocaeli University has been completed after the application for Ethical Committee approval and Ethical approval (100840098–6040.010.01-E.65168) has been given by Istanbul Medipol University Ethical Committee for Clinical Studies.

In the first stage of the study, it was planned to select patients with major depression as a result of psychiatric evaluations. The reason for this was to ensure that the patient group was homogeneous, to minimize the confounding effect of different factors, and because the most intensive use of the drugs in question is used in this patient group. Since there was no suggestion that SSRI and SNRI use made a difference in the study, homogeneity in this regard was ignored and it was important to select a similar patient group. Patients have been examined and diagnosed by qualified specialists in the psychiatry department with four years of professional experience as of the date of the study. The specialists were also trained physicians familiar with DSM-5 diagnostic criteria. Participants were recruited from a primary care practice. The inclusion criteria were patients who were older than 18 years of age, had not used any antidepressant during the six months prior to enrollment, were not currently taking any other medication, were systemically healthy, and were willing to sign the consent form.

After applying the inclusion criteria, patients were recruited based on a structured clinical interview for the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders - Fifth Edition (DSM-5). The 17-item, clinician-administered Hamilton Depression Rating Scale (HAMD) was administered

to depressed outpatients (Morriss et al. 2008; Rohan et al. 2016). Two expert raters independently scored each patient in the same interview. Only patients with equal scores from the two raters were included in the sample. The total 17-item HAMD score ranges from 0 to 52, with higher scores representing more severe depression. In this study, the 17-item HAMD cut-off points were defined as follows: >24 = severely depressed; 17-23 = moderately depressed; 8-16 = mildly depressed; and not depressed = 0-7 (Zimmerman et al. 2013). Based on these scores, moderately depressed patients were selected for the sample and prescribed SSRIs or SNRIs according to the judgment of expert psychiatrists. The dosage of the drugs was in accordance with the manufacturer's instructions.

Patients were then referred to Medipol University Faculty of Dentistry, Department of Oral and Maxillofacial Surgery for bruxism evaluation. Patients who did not attend scheduled follow-ups and patients diagnosed with TMD prior to antidepressant use were excluded to avoid confusion about the cause of TMD symptoms (Figure 1). The maxillofacial surgeons in this part of the study were experts with seven years, four years, and 23 years of professional experience, respectively, as of the date of the study.

The diagnosis of TMD was based on the DC/TMD form, physical examination, and patient feedback. Bruxism has been investigated using a self-assessment questionnaire and physical examination. Intraoral examination of the soft tissues and dental and extraoral examination of the masticatory muscles were performed by the same oral, dental, and maxillofacial surgeon by inspection and palpation according to the DC/TMD criteria. These examinations and patient feedback were used to diagnose awake bruxism. The fact that patients do not have TMD does not mean that they will not have Bruxism. It was also planned to evaluate the difference in Bruxism levels between T1 and T4. The questionnaire was related to the patients' clinical symptoms and complaints. The questions aimed to assess the presence of sleep bruxism and were based on the recommendations of Winocur et al. and the diagnostic criteria of the American Academy of Sleep Medicine (Winocur et al. 2011; Ito and Inoue 2015).

The questionnaire was applied to the patients during the first week of antidepressant treatment (T1) before any possible side effect can be experienced, and after the fourth week (T2), which is the optimal time of onset of side effects (Rajan and Sun 2017; Sabuncuoglu et al. 2009; Garrett and Hawley 2018). It consisted of four open-ended questions related to the demographic information, names of antidepressant medications, and ten questions that the respondents were asked to answer as yes/no. Specifically, the questions for the diagnosis of sleep bruxism aimed to enquire about the common symptoms experienced in the morning and the patient's awareness of the same based on the testimony of a sleep partner (Table 1). Diagnosis of sleep bruxism was confirmed if the respondent had a positive answer for questions 1 and /or 2, in addition to at least one positive response (yes) to a symptom listed in question 3.

Table 1: Questionnaire

Demographics & Questions	
Sex & Age:	
Systemic disease & related medication:	
Name of antidepressant used & duration:	
1	Are you aware, or has anyone heard you grinding your teeth frequently during sleep? (Yes/No)
2	Are you aware that your dentition is worn down more than it should be? (Yes/No)
3	Are you aware of any of the following symptoms upon awakening? (Yes/No):
Sensation of fatigue, tightness or soreness of your jaw?	
Feeling that your teeth are clenched or that your mouth is sore?	
Aching of your temples?	
Difficulty in opening your mouth wide?	
Feeling tension in your jaw joint on waking up & a feeling like you need to move your lower jaw to release it? (Yes/No)	
Hearing or feeling a ''click'' in your jaw joint on waking up, which disappears later? (Yes/No)	

IBM SPSS (Number Cruncher Statistical System) 2007 (Kaysville, Utah, USA) has been used to analyse the data. Descriptive statistical methods (mean, standard deviation, median, frequency, ratio, minimum, and maximum values) have been applied. Mann-Whitney U Test has been performed for a two-group comparison of quantitative data with non-normal distribution, and the Kruskal Wallis test has been used for comparing three or more groups.

As a result of the Shapiro-Wilks test, the kurtosis value of the dataset was found to be 1.146 and the skewness value was 0.857. Tabachnick and Fidell's study reported that values between -1.5 and +1.5 are appropriate for the assumption of normality (Tabachnick & Fidell 2013). Subsequently, descriptive analyses were carried out, taking into account that the dataset was appropriate for a normal distribution.

The comparison of T1 and T2 time intervals has been performed using Paired Sample t-test for parameters with normal distribution during follow-up. Wilcoxon Signed Rank Test for the parameters that were not normally distributed during follow-up was used. A p-value less than 0.05 was considered statistically significant, while a p-value less than 0.01 was considered highly statistically significant. The sample size of the study (n=150) has been calculated with 95% power, 0.5% margin of error and an effect size of 0.27.

RESULTS

In the study, 150 participants have been participated. Twenty-five patients were excluded from the study since they could not be properly followed up. The remaining 125 patients completed the follow-up and have been included in the data analysis (Figure 1). On the other hand, patients did not change their medications during the study. Demographic data of the participants are shown in Figure 2. Of the included patients, 84% (n = 105) were prescribed the SSRIs paroxetine, escitalopram, sertraline and fluoxetine, and 16% (n = 20) were prescribed the SNRIs duloxetine and venlafaxine (Table 2).

Table 2: Type of antidepressants & active ingredients

		n	%
Type of Antidepressants	SSRIs	105	84
	SNRIs	20	16
Active Ingredients	Paroxetine	18	14.4
	Duloxetine	13	10.4
	Escitalopram	31	24.8
	Sertraline	32	25.6
	Fluoxetine	24	19.2
	Venlafaxine	7	5.6

Only 32.8% of participants showed symptoms of bruxism during sleep and 45.6% showed symptoms of bruxism during awake time at the start of medication. In the fourth week of treatment, these rates increased to 40% and 54.4%, respectively (Figure 3). This increase has not been concluded to be statistically significant.

The scores for the presence of awake and sleep bruxism in the first and fourth weeks of antidepressant use were compared between genders (Table 3). In the first

Table 3: Presence of bruxism according to sex

		Male		Female		p
		n	%	n	%	
First Week (T1)	Sleep Bruxism	17	45,9	24	27,3	0,042*
	Awake Bruxism	16	43,2	41	46,6	0,778
Fourth Week (T2)	Sleep Bruxism	16	43,2	34	38,6	0,631
	Awake Bruxism	19	51,4	49	55,7	0,749

[b]*Pearson Chi-Square *p<0,05*

Table 4: Presence of bruxism according to antidepressant type

		Paroxetine (SSRI)		Duloxetine (SNRI)		Escitalopram (SSRI)		Sertraline (SSRI)		Fluoxetine (SSRI)		Venlafaxine (SNRI)		[b]*p*
		n	%	n	%	n	%	n	%	n	%	n	%	
First Week (T1)	Sleep Bruxism	5	27,8	4	30,8	11	35,5	11	34,4	9	37,5	1	14,3	0,889
	Awake Bruxism	9	50,0	7	53,8	13	41,9	15	46,9	15	46,9	2	28,6	0,913
Fourth Week (T2)	Sleep Bruxism	11	61,1	9	69,2	11	35,5	11	34,4	7	29,2	1	14,3	0,038*
	Awake Bruxism	11	61,1	8	61,5	15	48,4	20	62,5	11	45,8	3	42,9	0,703

[b]*Pearson Chi-Square **p<0,01 *p<0,05*

week of medication initiation, 45.9% of male patients and 27.3% of female patients showed symptoms of sleep bruxism. The incidence was higher in male patients ($p = 0.042$; $p < 0.05$).

When SSRIs and SNRIs were compared, paroxetine in the SSRI group and duloxetine in the SNRI group caused an increase in the presence of sleep bruxism ($p = 0.013$; $p < 0.05$) (Table 4). Escitalopram, fluoxetine and sertraline in the SSRI group and venlafaxine in the SNRI group had no significant effect on awake or sleep bruxism.

Due to the difference in the number of patients using SSRIs (105) and SNRIs (20), partial eta squared calculation was performed to calculate the effect size. The $\eta p^2 =$ SS between / SS total. + SS error formula has been used in the calculation and the effect size was determined as 0.437.

DISCUSSION

Based on the knowledge available, this is the first study to evaluate the time of onset (awake/sleep time) and the presence of bruxism, especially in the early phase of SSRI and SNRI use. The authors hypothesized that SSRIs and SNRIs may be risk factors for early bruxism. This hypothesis has been partially confirmed by the finding that some SSRIs and SNRIs cause early stage bruxism. Paroxetine, an SSRI, and duloxetine, an SNRI, increased the symptoms of early bruxism, whereas escitalopram and fluoxetine, SSRIs, and venlafaxine, SNRIs, did not cause such effects. Paroxetine and duloxetine caused this effect only for sleep bruxism but did not affect awake bruxism.

Lobbezoo et al. reported in a study on SSRIs and SNRIs that bruxism may be associated with SSRI use (Lobbezoo et al. 2001). Since these drugs are widely used today, SSRI and SNRI-induced bruxism is one of the critical research areas. Garret et al. reported a possible association of sertraline and fluoxetine with secondary bruxism (Garrett and Hawley 2018). Fitzgerald stated that sertraline, paroxetine and fluoxetine triggered bruxism in a case series of 6 patients (Fitzgerald and Healy 1995). In a similar report, Sabuncuoglu et al. also reported that sertraline and fluoxetine caused bruxism symptoms four weeks after starting the medication (Sabuncuoglu et al. 2009). Various cases of bruxism caused by SSRIs sertraline, fluoxetine and paroxetine have been previously reported (Akbaş and Bilgiç 2018; Romanelli et al. 1996; Soyata and Oflaz 2015; Zandifar, et al. 2018).

Similar to the findings of the present study, Ellison et al. reported that sertraline, fluoxetine and escitalopram did not cause secondary bruxism, but paroxetine may cause it. In addition, duloxetine and venlafaxine can also induces bruxism (Behr et al. 2012). Some other case reports and systematic reviews have noted bruxism as a side effect of duloxetine and venlafaxine (Xue et al. 2016; Melo et al. 2018;Kuloglu et al. 2010; Sahin Onat and Malas 2015). On the contrary, SNRIs lead to a selective permeability for serotonin and noradrenaline. Montgomery has proved that this effect of venlafaxine on serotonin receptors is about three times greater than duloxetine (Montgomery 2008). In one case report, venlafaxine-induced bruxism was resolved by switching to duloxetine (Chang et al. 2011). However, in our study, duloxetine has been found as an agent inducing bruxism, but venlafaxine has not been found.

Studies on the onset of secondary bruxism due to SSRIs and SNRIs reported a similar period after starting the drug. It has been reported that bruxism may occur from the second week after the serotonin effect is seen (Garrett and Hawley 2018). There is no data on whether the onset time is related to the drug type and dose. There is also no consensus on the relationship between SSRI or SNRI-induced bruxism and age or gender. In the present study, no relationship has been found between age and secondary bruxism symptoms. However, there has been a difference in the occurrence of sleep bruxism secondary to SSRIs and SNRIs in males; the rates of this bruxism have been found to be higher after four weeks of medication use.

Currently, a consensus does not exist on how to reduce bruxism symptoms caused by SSRIs and SNRIs; however, some case reports suggest reducing the dose or discontinuing the medication (Kuloglu et al. 2010; Rajan and Sun 2017; Milanlioglu 2012). Rajan et al. reported that bruxism symptoms improved on the third day when they reduced venlafaxine from 225 mg/day to 187.5 mg/day (Rajan and Sun 2017). However, in light of the current literature, it is not known whether secondary bruxism symptoms are permanent. Milanlioglu et al. observed that bruxism symptoms decreased after discontinuation of paroxetine; however, secondary bruxism caused by SSRIs was persistent even after drug treatment was discontinued (Milanlıoglu 2012). One of the treatment options to eliminate SSRI or SNRI-induced bruxism is to switch to drug derivatives with different mechanisms of action. For this purpose, buspirone, aripiprazole, quetiapine, gabapentin, amitriptyline, mirtazapine and tandospirone have been tried (Zandifar et al. 2018; Soyata and Oflaz 2015; Akbaş and Bilgiç 2018; Sahin Onat and Malas 2015;Bostwick and Jaffee 1999; Khosravi 2020; Kishi 2007; Oulis et al. 2012).

Bruxism is considered to have a complex and multifactorial etiology that affects multiple physiological processes (Klasser et al. 2015). Studies report that stress,

personality and psychological state are common predisposing factors. Anxiety and depression have been reported to be mainly associated with awake bruxism. In an epidemiologic study, no clear association was found between sleep bruxism confirmed by polysomnography and anxiety or depression (Maluly et al. 2013). In addition, many studies aiming to assess the role of psychological stress in the etiology of bruxism have been reported to be unreliable. Therefore, anxiety and depression cannot be considered as direct sources of bruxism (Feu et al. 2013).

As limitations of the presented study, we can mention the relatively low number and disproportionate cases. Secondly, both depression and antidepressant use to treat depression could be considered as contributing factors for bruxism which can cause confusion.

CONCLUSION

Findings of the current study suggest that certain antidepressants may contribute to the emergence of sleep bruxism, warranting further investigation into potential underlying mechanisms and management strategies. Our study showed that some SSRIs and SNRIs caused early-stage secondary bruxism. Paroxetine (SSRI) and duloxetine (SNRI) only increased sleep bruxism among the evaluated drugs. None of the antidepressants evaluated reduced current bruxism. In the opinion of the authors, more standardized and reliable diagnostic tools and criteria for bruxism should be developed. Furthermore, more randomized controlled and longitudinal clinical trials are needed to assess whether SSRIs and SNRIs with similar mechanisms of action show a similar effect or the long-term effect of secondary bruxism. To prevent the risk of bruxism-induced TMD in the future, the need for detailed patient assessment before prescribing SSRIs and SNRIs should not be disregarded.

Ethical Considerations: Does this study include human subjects? YES
Authors confirmed the compliance with all relevant ethical regulations.
Conflict of interest: No conflict of interest
Funding sources: The authors received no funding from an external source.
Authors Contributions: dr. Ipek Necla Guldiken – study design, first draft, approval and final version, statistical analysis. dr. Begüm Elbir – data collection, first draft, approval of the final version, statistical analysis. dr. Andrei Nalimov – data collection, first draft, approval of the final version. Prof. Çağri Delilbaşi – study design, first draft, approval of the final version

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